Case Report

Successful treatment of non-convulsive status epilepticus diagnosed using bedside monitoring by a combination of amplitude-integrated and two-channel simplified electroencephalography

Satoshi Egawa, Toru Hifumi, Kenya Kawakita, Arisa Manabe, Hikari Matumura, Tomoya Okazaki, Hideyuki Hamaya, Natuyo Shinohara, Hajime Shishido, Koshiro Takano, Yuko Abe, Masanobu Hagiike, and Yasuhiro Kuroda

Emergency Medical Center, Kagawa University Hospital, Miki, Kagawa, Japan

Case: A 66-year-old man developed disturbed consciousness and right hemiparesis with transient convulsions in the right arm. Bedside monitoring using a combination of amplitude-integrated electroencephalography and two-channel simplified electroencephalography revealed intermittent episodes of 1–3 Hz δ waves lasting for approximately 5 min, consistent with non-convulsive status epilepticus. Fosphenytoin (22.5 mg/kg/day) and levetiracetam (1,000 mg) prevented right arm convulsions but did not restore consciousness. The two-channel simplified electroencephalography also showed an intermittent periodic δ wave pattern in the Fp1-C3 channel. Conventional electroencephalography revealed a polymorphic δ activity that was abolished by 2.5 mg diazepam, thus confirming the diagnosis of non-convulsive status epilepticus.

Outcome: The patient recovered completely with the antiepileptic drug combination.

Conclusion: Immediate initiation of bedside monitoring using amplitude-integrated electroencephalography and two-channel simplified electroencephalography allows early detection of non-convulsive status epilepticus in patients with disturbed consciousness, which considerably improves the prognosis.

Key words: Epilepsy, non-convulsive status epilepticus, electroencephalography, diazepam

INTRODUCTION

NON-CONVULSIVE STATUS EPILEPTICUS (NCSE) is a relatively common condition among patients with unexplained altered mental status, with a prevalence of 8–37%.[1,2] It must be diagnosed and treated rapidly to avoid significant morbidity and mortality.[3] A retrospective study on 100 NCSE patients identified the mortality rate as 18%.[3] Non-convulsive status epilepticus is currently diagnosed by continuous electroencephalography (EEG), which requires trained personnel, thereby delaying treatment.[4] Accordingly, there is a pressing need for a simpler method that acute care physicians could use to evaluate patients with clinical symptoms of NCSE. Recently, a non-invasive monitoring system combining amplitude-integrated EEG (aEEG) and two-channel simplified EEG (sEEG) was designed to monitor cerebral activities using bedside monitoring. The aEEG system is widely used to predict the outcome of hypoxic ischemic encephalopathy to therapeutic hypothermia in neonates[5] and adults.[4] To our knowledge, this approach (Fig. 1, left panel) has not been used to diagnose NCSE. Here we report a case of NCSE diagnosed on the first day of admission using the bedside monitoring system using a combination of aEEG and sEEG. The diagnosis was confirmed by conventional EEG and benzodiazepine responsiveness, and the patient was successfully treated. This case supports the usefulness of this approach for the early diagnosis of NCSE in patients with disturbed consciousness.

Corresponding: Toru Hifumi, MD, Emergency Medical Center, Kagawa University Hospital,1750-1 Ikenobe, Miki, Kita, Kagawa 761-0793, Japan. E-mail: hifumitoru@gmail.com.
Received 29 May, 2015; accepted 15 Jul, 2015; online publication 27 Aug, 2015

CASE REPORT

A 66-YEAR-OLD MAN DEVELOPED a disturbed state of consciousness while undergoing chemotherapy for pancreatic cancer at a local hospital. He had a medical

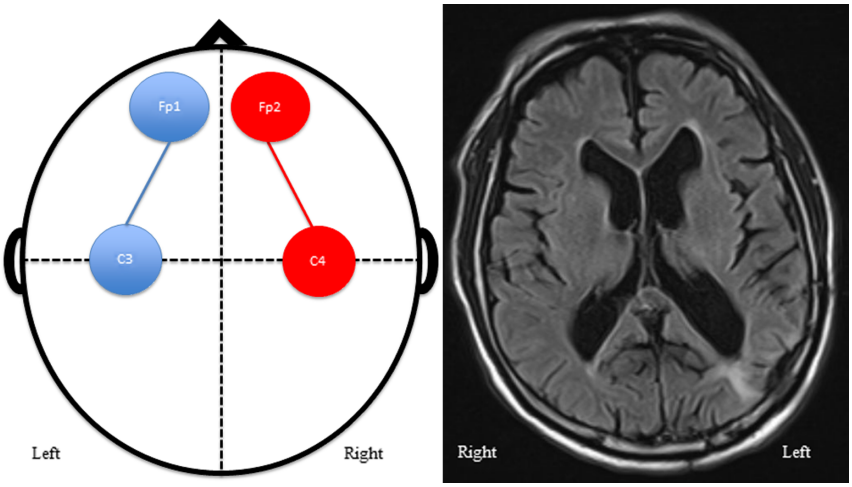

Fig. 1. Baseline cerebral status and diagnostic strategy. Left panel, configuration of the bedside two-channel amplitude-integrated electroencephalography device: FP1-C3 and FP2-C2. Right panel, magnetic resonance imaging with fluid attenuation inversion recovery in a 66-year-old man diagnosed with non-convulsive status epilepticus. The high-density area located in the left occipital lobe reflects a previous surgery for meningioma resection.

history of left occipital lobe meningioma, which was removed 2 years prior to his admission. His Glasgow Coma Scale score was 8/15 (E1V2M5). This test revealed right hemiparesis with transient convulsions in the right arm. Laboratory data indicated normal hepatic and kidney functions with mildly elevated C-reactive protein (1.73 mg/dL; normal range, <1.0 mg/dL) and white blood cell count (11,930/μL; normal range, 4,500–10,000/μL). Magnetic resonance imaging detected no abnormality, except the consequences of the surgery on the left occipital lobe (Fig. 1, right panel). The patient was given 250 mg phenytoin and transferred to our hospital for further investigation of the altered mental status.

On admission, the vital patient's signs were stable, but his consciousness level remained disturbed (E2V2M5). He had right hemiplegia with a manual muscle testing score of 3/5 and sporadic convulsions in the right arm. Therefore, bedside monitoring was initiated in the intensive care unit (ICU) using a combination of aEEG (Fig. 1, left panel) and two-channel sEEG. The acute care physicians noticed intermittent episodes of typical 1–3 Hz δ epilepsy waves (Fig. 2) lasting approximately 5 min each time, a pattern consistent with a diagnosis of NCSE. Therefore, the recommended treatment for NCSE was initiated on the first day of admission, a combination of fosphenytoin (fPHT; 22.5 mg/kg/day) and levetiracetam (1,000 mg). The treatment prevented right arm convulsions but did not improve his consciousness level.

Amplitude-integrated EEG also showed an intermittent periodic δ wave pattern in the Fp1-C3 channel. Therefore, neurological specialists were consulted on day 1 of admission, who decided to carry out the 30-min conventional EEG on the same day. This approach revealed polymorphic δ activity characterized by spikes and slow waves in P3-A1

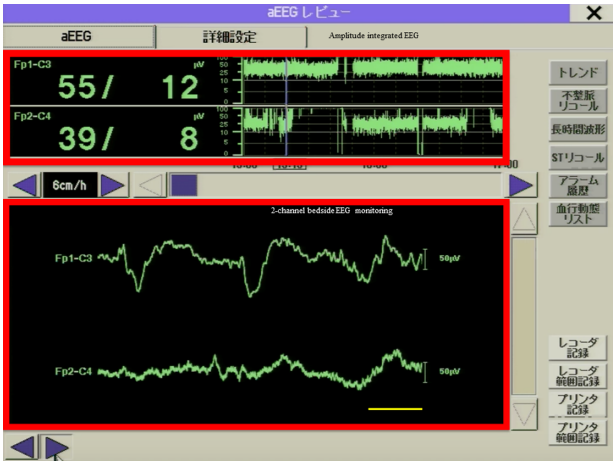

Fig. 2. Image capture of the amplitude-integrated electroencephalography (aEEG) screen showing δ waves with occasional spikes (Fp2-C4), as well as Fp1-C3 waves, in a 66-year-old man diagnosed with non-convulsive status epilepticus. Yellow bar = 1 s.

and O1-A1 (Fig. 3, left panel). And it accompanies evolution. These waves were practically abolished by treatment with 2.5 mg diazepam (Fig. 3, right panel) which confirmed the diagnosis of NCSE. Therefore, the patient was treated for a further 3 days with a combination of levetiracetam (1,000 mg/day) and low-dose fPHT (7.5 mg/kg/day). On day 4, the dose of fPHT was tapered based on aEEG and sEEG traces and clinical symptoms; the patient's consciousness level had improved to E4V5M6. On day 7, a second 30-min conventional EEG indicated that the polymorphic δ activity had disappeared and that the traces had become normal. On day 18, the consciousness level of the

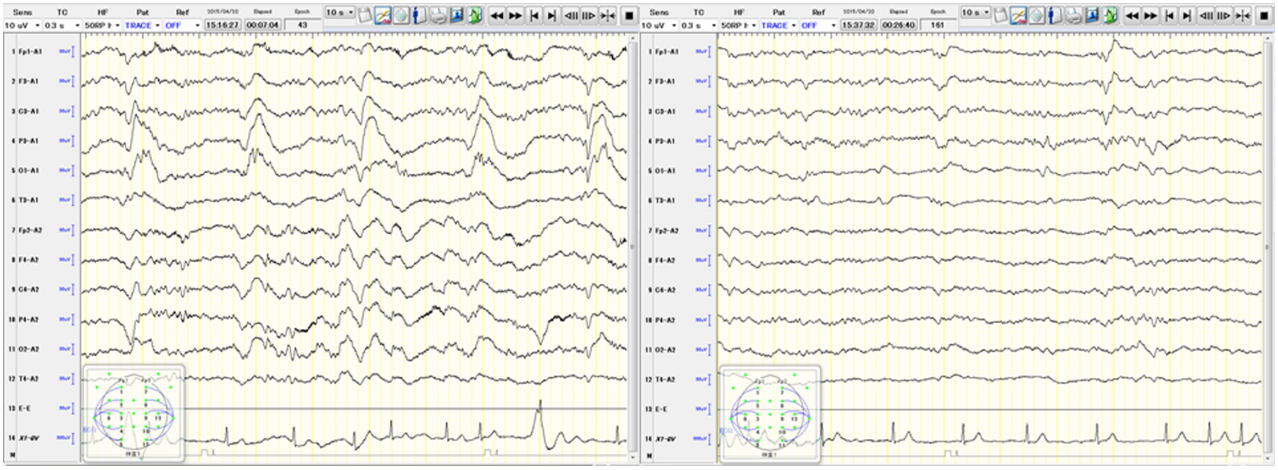

Fig. 3. Conventional electroencephalography carried out on a 66-year-old man diagnosed with non-convulsive status epilepticus. Left panel, continuous δ waves in P3-A1 and O3-A1 lasting over 5 min. Right panel, suppressive effect of benzodiazepine therapy on the δ waves.

patient was restored to normal, and he was discharged from our hospital.

DISCUSSION

THE PRESENT REPORT shows that the bedside monitoring system using a combination of aEEG and sEEG can be used for the early diagnosis of NCSE in the case of patients with disturbance in consciousness, and to monitor their treatment responses. The clinical application of sEEG has only recently been discussed; therefore, no definitive data are available in clinical practice.[6] Whereas conventional EEG only provides data over a period of 30 min and requires highly trained neurophysiologists, the aforementioned approach allows continuous monitoring by acute care physicians as soon as the patient is assigned to a hospital bed.[7] In the current case, NCSE was suspected, but could not be confirmed by the sEEG system because of the inherently low sensitivity of this system. The single-channel EEG detected 78% of seizures and 94% of infants with seizures.[8] Furthermore, the sensitivities of seizure detection by sEEG were directly correlated with the number of leads. Using seven leads[9] and four leads[10] yielded sensitivities of 93% and 68%, respectively. Single-channel EEG yields a sensitivity of 40%.[11] The application of bedside sEEG to the diagnosis of NCSE may be limited to patients particularly at risk of developing disturbed consciousness following generalized convulsive status, recurrent coma, facial myoclonus, rapid involuntary eye movements, or aphasia.[10]

The interpretation of aEEG and sEEG traces requires some training to recognize the patterns indicative of NSCE and are divided into the following groups: rhythmicity, spike and wave, and periodicity.[12] Furthermore, all types of EEG are affected by the following artifacts: mechanical ventilation, suction, and blinking. Video-EEG monitoring would be the ultimate modality to diagnose NCSE in the ICU.[13] This equipment is currently not available in most ICUs of Japan. The aEEG and sEEG monitoring should be introduced in the ICU settings to improve the detection of abnormal brain activities indicative of conditions such as NCSE. The Kagawa University Hospital (Kagawa, Japan) currently offers appropriate training for bedside aEEG and sEEG systems.

In conclusion, this case supports the usefulness of the bedside monitoring system using a combination of aEEG and sEEG for the early diagnosis of NCSE, which can then be confirmed by conventional EEG and benzodiazepine responsiveness. This system improves the chances of early detection of NCSE by any doctor, who may then consult neurologists to confirm the diagnosis and prescribe the appropriate treatment.

CONFLICT OF INTEREST

NONE.

REFERENCES

1 Zehtabchi S, Abdel Baki SG, Omurtag A *et al*. Prevalence of non-convulsive seizure and other electroencephalographic abnormalities in ED patients with altered mental status. Am. J. Emerg. Med. 2013; 31: 1578–82.

2 Towne AR, Waterhouse EJ, Boggs JG *et al*. Prevalence of nonconvulsive status epilepticus in comatose patients. Neurology 2000; 54: 340–5.
3 Shneker BF, Fountain NB. Assessment of acute morbidity and mortality in nonconvulsive status epilepticus. Neurology 2003; 61: 1066–73.
4 Rundgren M, Rosen I, Friberg H. Amplitude-integrated EEG (aEEG) predicts outcome after cardiac arrest and induced hypothermia. Intensive Care Med. 2006; 32: 836–42.
5 Horn AR, Swingler GH, Myer L *et al*. Early clinical predictors of a severely abnormal amplitude-integrated electroencephalogram at 48 hours in cooled neonates. Acta Paediatr. 2013; 102: e378–84.
6 Friberg H, Westhall E, Rosen I *et al*. Clinical review: Continuous and simplified electroencephalography to monitor brain recovery after cardiac arrest. Crit. Care 2013; 17: 233.
7 Shah NA, Wusthoff CJ. How to use: Amplitude-integrated EEG (aEEG). Arch. Dis. Child. Educ. Prac. Ed. 2015; 100: 75–81.
8 Shellhaas RA, Soaita AI, Clancy RR. Sensitivity of amplitude-integrated electroencephalography for neonatal seizure detection. Pediatrics 2007; 120: 770–7.
9 Karakis I, Montouris GD, Otis JA *et al*. A quick and reliable EEG montage for the detection of seizures in the critical care setting. J. Clin. Neurophysiol. 2010; 27: 100–5.
10 Young GB, Sharpe MD, Savard M *et al*. Seizure detection with a commercially available bedside EEG monitor and the subhairline montage. Neurocrit. Care 2009; 11: 411–6.
11 Nitzschke R, Muller J, Engelhardt R, Schmidt GN. Single-channel amplitude integrated EEG recording for the identification of epileptic seizures by nonexpert physicians in the adult acute care setting. J. Clin. Monit. Comput. 2011; 25: 329–37.
12 Hirsch LJ, LaRoche SM, Gaspard N *et al*. American Clinical Neurophysiology Society's standardized critical care EEG terminology: 2012 version. J. Clin. Neurophysiol. 2013; 30: 1–27.
13 Dericioglu N, Yetim E, Bas DF *et al*. Non-expert use of quantitative EEG displays for seizure identification in the adult neuro-intensive care unit. Epilepsy Res. 2015; 109: 48–56.